Diabetic Ketoacidosis

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KEYWORDS

• Diabetic ketoacidosis • Euglycemic diabetic ketoacidosis • Hyperglycemia

KEY POINTS

- Diabetic ketoacidosis is a common life-threatening emergency that requires prompt diagnosis and treatment.
- Inciting factors to consider include: infection, medication nonadherence or changes, intoxication, pregnancy, and myocardial or cerebral infarction.
- Euglycemic DKA requires a high index of suspicion and should be considered in patients with risk factors (ie, pregnancy, starvation, and SLGT-2 inhibitor use).
- Treatment should begin with intravenous isotonic or balanced fluids. Fluids can be started with the initial elevated glucometer reading of hyperglycemia before the remainder of the laboratory tests are back.
- The insulin infusion should not be stopped until the anion gap is closed, and the metabolic acidosis is resolved, as evidenced by normalization of the bicarbonate level.

INTRODUCTION

Diabetic ketoacidosis (DKA) is a common hyperglycemic emergency and acute life-threatening complication of diabetes mellitus. Per the Centers for Disease Control and Prevention, more than 37 million Americans are living with diabetes, and approximately 8.5 million are presently undiagnosed.[1] In 2017, the cost of diabetes in the United States was estimated at $327 billion, with $237 billion spent on direct patient care.[2] Characteristically, DKA is associated with type 1 diabetes but is also seen in patients with type 2 diabetes, especially those presenting with an inciting event, which is discussed in detail later. It is associated with uncontrolled diabetes and is known to have high morbidity and mortality.[3] A report published in 2018 described a steady increase in DKA hospitalizations from 2009 to 2014 but a decrease in the

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in-hospital case mortality rate from 1.1 to 0.4.[4] Nonetheless, DKA is often encountered by emergency physicians (EP), and it requires a timely evaluation, diagnosis, and treatment.

DIAGNOSIS

Clinical Diagnosis of Diabetic Ketoacidosis

The importance of promptly recognizing DKA in the emergency department (ED) cannot be understated, because it is a life-threatening emergency requiring timely management. The development of DKA typically happens quickly and may present with minimal signs and symptoms.[5] Clinically, patients may have a variety of symptoms on presentation, which include polyuria, polydipsia, weight loss, abdominal pain, nausea, vomiting, dehydration, weakness, dyspnea, and mental status changes.[5,6] A patient's mental status can range from full alertness to lethargy, stupor, or coma.[5,6] Physical examination findings may include signs of dehydration, such as poor skin turgor and dry mucous membranes.[5,6] Tachypnea or Kussmaul breathing, characterized as rapid deep breathing, are frequently described as a tell-tale sign of DKA, as is the presence of a "fruity odor" of the breath.[6] Other findings may include tachycardia and hypotension, especially in a patient with severe dehydration and a concurrent infection leading to sepsis.[5] Taking a careful patient history and looking for risk factors for DKA can allude to the diagnosis. Consider other historical factors, such as medication history and adherence, infectious symptoms, and history of substance use.[7]

It is essential to recognize and consider the likelihood of precipitating factors in the development of DKA, and it is essential to consider why the patient is presenting with DKA. Sometimes referred to as the five "I's," infection, infarction (myocardial, cerebral), infant (pregnancy), indiscretion (dietary or intoxication), and insulin (noncompliance) are often cited as common triggers for DKA.[8] One must also consider other factors, including medication discontinuation, undertreatment, pancreatitis, thyrotoxicosis, trauma, cocaine use, a new diagnosis of diabetes, and other metabolic disorders.[6,8,9]

Infection is arguably the most common precipitating factor involved in the development of DKA.[5] A single-center study revealed that medication nonadherence or discontinuance was the leading cause of DKA in inner-city African-American patients.[10] Patients who are started on certain medications, such as steroids, may also be at increased risk. Other medications thought to precipitate DKA include thiazides, sympathomimetics, pentamidine, and antipsychotics.[5,11,12]

Laboratory Work-up/Imaging

The diagnostic criteria for DKA are not well defined, but hyperglycemia, anion gap metabolic acidosis (AGMA), and ketonemia are classic abnormalities.[8] Patients should undergo an initial laboratory work-up, which includes plasma glucose level, complete blood counts with differential, chemistry (to include potassium, sodium, magnesium, and phosphorous), blood urea nitrogen, creatinine, anion gap, blood gas, blood cultures, lactate, urinalysis, urine ketones, and evaluation of ketonemia if available.[5,6] Possible laboratory findings are detailed next. An electrocardiogram should be obtained and may identify signs of ischemia or electrolyte abnormalities, notably hyperkalemia or hypokalemia.[7]

Imaging should be targeted to the patient's presentation. It may include chest radiographs or computed tomography of the head, chest, or abdomen/pelvis depending on the patient's specific presentation. Although abdominal pain is a common

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presentation in DKA, not all of these patients require imaging. However, there should be a low threshold to obtain imaging if the patient is febrile, hypotensive, or has a concerning abdominal examination with peritoneal signs.

Laboratory Findings

Common initial laboratory abnormalities include the following:

- Glucose: Usually is elevated greater than 250 mg/dL but can range from normoglycemia to levels greater than 600 mg/dL.[5] Approximately 2.6% to 3.2% of patients present with euglycemia or glucose levels less than 250 mg/dL.[13]
- Sodium: Patients commonly present with a pseudohyponatremia caused by the osmotic pull generated by hyperglycemia.[5] Hypernatremia and even normal sodium levels indicate profound free water losses.[5] Although traditionally EPs have been taught to correct for this by adding 1.6 mmol/dL sodium per 100 mg/dL glucose greater than 100 to the reported sodium value, a more recent paper has shown that the correction factor should be 2.4 mmol sodium per 100 mg glucose greater than 100.[14]
- Potassium: May be elevated because of intracellular potassium shifts in the setting of acidosis and insulin deficiency.[7] Low potassium levels represent a severe total body potassium deficiency and should be carefully, but aggressively corrected to avoid cardiac dysrhythmia.[7]
- Magnesium: Usually low and requires replacement therapy along with potassium.[7]
- Blood urea nitrogen, and creatinine: Elevations in both are seen in DKA and are often caused by hypovolemia and a prerenal acute kidney injury.
- Serum pH: A pH less than 7.3 is consistent with acidosis/DKA. It is possible to have a normal or even raised pH if the patient has another concomitant metabolic or respiratory alkalosis.[7] There is no need to obtain an arterial blood gas unless one has concerns with oxygenation; a venous blood gas is just as accurate.[15]
- Bicarbonate: Levels are expected to be low but are influenced by other conditions, such as underlying chronic metabolic alkalosis or chronic hypercapnia because of chronic obstructive pulmonary disease, which results in a corrective metabolic alkalosis. An astute EP looks for triple base disorders in patients with underlying lung disease.
- Anion gap: A normal anion gap is 12. The anion gap is elevated because of the presence of ketones.[7] The anion gap is calculated using the provided laboratory values. The pseudohyponatremia that is present should not be corrected before determining the anion gap.[16]
- Ketones: Three ketones are known to accumulate in DKA: (1) acetoacetic acid, (2) acetone, and (3) β-hydroxybutyrate. β-Hydroxybutyrate is a key diagnostic feature in DKA. Depending on the hospital laboratory capability, these laboratory studies can be sent; however, they are not always available for timely testing or essential for diagnosis/treatment.
- White blood cell (WBC) count: A leukocytosis in the range of 10,000 to 15,000 is often seen in patients with DKA without an active infection process, but WBC greater than 25,000 or a bandemia greater than 10% should prompt an evaluation for infection.[5]

Differential Diagnosis

Hyperglycemia has a myriad of causes. It is crucial to consider the possibility of a hyperosmolar hyperglycemic state when patients with type 2 diabetes present with

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hyperglycemic crisis.[17] Consider other reasons for an AGMA, such as starvation ketosis, alcoholic ketosis, lactic acidosis, ethylene glycol intoxication, methanol intoxication, salicylate ingestion, other ingestions, uremia, and acute renal failure.[17,18] Clinically, a thorough history is essential in distinguishing between these differentials, and the addition of laboratory tests to evaluate for other ingestions may be helpful.

Euglycemic Diabetic Ketoacidosis

As the name suggests, euglycemic diabetic ketoacidosis (EDKA) is ketoacidosis with a normal blood glucose concentration.[19] It is estimated that 2.6% to 3.2% of patients with DKA present with EDKA (glucose level <250 mg/dL).[13] Although uncommon, appreciation of this entity is of supreme importance because it is a life-threatening emergency that can result in delays in care and significant morbidity if not recognized.[19] It is difficult to diagnose and requires a high index of suspicion to be recognized. Several different etiologies for developing EDKA have been discussed in the literature. These include starvation, pregnancy, administration of insulin given on route to the hospital, inhibition of gluconeogenesis, sepsis, insulin pumps, acute pancreatitis, and such medications as sodium-glucose cotransporter (SLGT-2) inhibitors.[19,20]

Since the advent of SGLT-2 in the treatment of diabetes, there have been cases of EDKA reported and recognized, resulting in a Food and Drug Administration alert in 2015.[21,22] Patients on SGLT-2 are estimated to have a seven-fold increased risk of developing acidosis.[23] SGLT-2 works by inhibiting the sodium-glucose cotransporter leading to glucose excretion into the urine.[24] The theory behind the pathophysiology of SGLT-2 in causing EDKA is multifactorial and still not fully understood. The action of these medications on the kidneys is proposed to cause ketone reabsorption during the excretion of glucose. In contrast, their action at the pancreas inhibits β cells leading to lipolysis also contributing to ketonemia.[25] Increases in glucagon and catecholamine levels via SGLT-2, which is exacerbated by dehydration, is also proposed as a mechanism in EDKA.[26]

Pregnancy is an identified risk for developing EDKA. Pregnant patients have a higher risk of developing DKA than the general population.[19] The physiologic changes of pregnancy are the precise bases that predispose them to EDKA, which includes a state of hypoinsulinemia and a relative state of insulin resistance, which develops in the second and third trimesters. Although EDKA may be challenging to diagnose, it should be considered in pregnant patients with nausea, vomiting, and unexplained acidosis.[19] Starvation or fasting acts to decrease glycogen stores and increases the risk of EDKA in patients with these risk factors.

Patients who develop EDKA may have various symptoms on presentation, including fatigue, abdominal pain, nausea, vomiting, shortness of breath, malaise, and decreased oral intake.[20,27] They may have symptoms similar to hyperglycemic DKA; however, they may also have a more insidious onset.[20] Their examination findings, like DKA with hyperglycemia, may include tachypnea, including Kussmaul breathing, tachycardia, and signs of dehydration (ie, decreased skin turgor), and delayed capillary refill.[20,27] Precipitating factors should also be considered for EDKA and are the same as DKA as detailed earlier including pregnancy, infection, and insulin dosing changes.[25] Patients who use insulin, including patients with insulin pumps, should be asked about the timing and amount of their last insulin bolus. A focused review detailing the risks and triggers for EDKA in the setting of SGLT-2 also described the risk of perioperative fasting. The medication's long half-life, and the risk of further ketonemia in starvation, increases the risk of EDKA.[27]

Making the diagnosis of EDKA is difficult. EPs should have a broad differential in any patient who presents to the ED with an AGMA and a normal blood glucose

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level.[19] It should be considered in patients with the previously mentioned symptoms, especially if they take an SGLT-2 or have other risk factors. A standard diagnostic approach is a suitable starting point, including serum pH, and blood and urine ketone measurements.[20,28,29] In EDKA, laboratory results reveal a blood pH of less than 7.3, increased anion gap, blood and urine ketones, and blood glucose less than 250 mg/dL.[19,26,27]

TREATMENT

Fluids

The treatment of DKA starts with fluid administration. This can be started before any laboratory values are known, and the patient is only known to be hyperglycemic. Most patients with DKA are profoundly dehydrated because of the osmotic diuresis that hyperglycemia causes and often need 4 to 6 L of volume repletion. Some guidelines recommend starting with normal saline 0.9%. However, the authors favor a balanced crystalloid solution (ie, lactated Ringers, plasmalyte) because this prevents the development of a hyperchloremic nonanion gap metabolic acidosis (NAGMA) that can result with normal saline. One study even showed a more rapid resolution of DKA when balanced crystalloids are used.[30] The disadvantage of using a balanced solution is that it is not available in premade solutions with additional potassium chloride. Patients can require 2 to 4 L of fluid boluses. Hypovolemia can increase insulin resistance by increasing the release of stress hormones (ie, cortisol, catecholamines), so it is important to get the patient to a euvolemic state. Once the patient has reached euvolemia following fluid bolus but the blood glucose remains greater than 300 mg/dL, balanced fluids should be continued but decreased to a rate of 150 mL/h to 200 mL/h. Once the blood glucose is less than 300 mg/dL and the anion gap is not yet closed, dextrose-containing fluid should be started in order for the insulin to be started or continued. Insulin is required to shut off ketogenesis and must be continued even if blood glucose levels decrease. The ongoing infusion of balanced fluids can be split to accommodate the addition of dextrose-containing fluids. Patients can receive dextrose 5% or dextrose 10% and a balanced solution using a two-bag system.[31] The patient can always receive more dextrose to maintain their blood glucose levels while receiving insulin.

If normal saline was used, remember that the NAGMA that is created can prolong the acidosis if it is not treated. Bicarbonate may need to be administered to correct the NAGMA. EPs should consider this when they notice that the anion gap has closed but that the patient's bicarbonate level has not improved.

Electrolytes

The profound diuresis that results from hyperglycemia is associated with several electrolyte disorders. Specifically, patients tend to be whole-body potassium and phosphate depleted. The serum sodium level may also seem artificially low because of the osmotic effect of the glycemia.

Whole-body potassium is low because of losses in the urine; however, the initial laboratory values may be elevated as the body tries to accommodate the metabolic acidosis. As hydrogen (H+) ions are transported into the cells, potassium is transported out to maintain cation balance. Therefore, the serum potassium level may be artificially elevated. Patients should start receiving potassium supplementation once the serum potassium is less than 4.5 mmol/dL. Before starting insulin, EPs must ensure potassium levels are greater than 3.3 mmol/dL. Because the effects of insulin are to drive glucose and potassium into the cells, the patients could become

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profoundly hypokalemic leading to cardiac arrhythmias and cardiovascular collapse if started too early. **Table 1** provides potassium supplementation recommendations.

Although total-body phosphate may be low and can lead to fatigue and decreased energy, this does not need to be replaced urgently. Attention should be on correcting and maintaining the potassium level.

Patients with significant hyperglycemia seem to be hyponatremic on routine laboratory studies. However, this is a laboratory abnormality caused by the dilution effect of glucose and the osmotic changes that occur in the serum. Although traditionally EPs have been taught to correct for this by adding 1.6 mmol/dL sodium per 100 mg/dL glucose greater than 100 to the reported sodium value, a more recent paper has shown that the correction factor should be 2.4 mmol/dL sodium per 100 mg/dL glucose greater than 100.[14] The most important thing to remember is that hyponatremia that corrects for hyperglycemia is not the cause of the patient's symptoms and does not need rapid correction with 3% normal saline. If the patient is hypernatremic, with a corrected sodium greater than 150 mmol/dL, some authors recommend selecting 0.45% normal saline as the balanced crystalloid once the patient is euvolemic.[32]

A basic metabolic profile should be obtained every 2 to 4 hours to monitor for hypokalemia, monitor the acidosis, and evaluate the metabolic anion gap.

Insulin

Although hyperglycemia can be corrected and treated with intravenous (IV) fluids, insulin is required to turn off ketogenesis and correct the metabolic acidosis. Once the potassium level is known and greater than 3.3 mEq/L, insulin is started. There is often debate about the best insulin administration route, which has gotten more attention recently because of the national regular insulin shortage seen in 2022. Studies have shown that IV and subcutaneous (SQ) insulin are effective in correcting DKA. However, in hypotensive patients or patients on vasoactive agents, the absorption of SQ insulin could be affected, so the IV route is preferred in these patients.

Numerous protocols exist for IV insulin and whether it should be given as a bolus followed by an infusion or if the infusion can just be started. The authors prefer not to use a bolus because of the increased risk of hypoglycemia that can result from this method. The goal is for the serum glucose to decline by about 100 mg/dL/h. However, an insulin bolus is helpful if there is a delay in getting the insulin infusion started (ie, need to request it from the pharmacy) or if the patient has severe acidosis,

Table 1
Potassium supplementation recommendations

Potassium Level	Supplementation Recommendation	Comments
<3.3 mEq/L	20 mEq/h IV[a] Some authors recommend up to 40 mEq/h with careful monitoring[37,38]	*Hold* Insulin infusion can exacerbate hypokalemia.
3.3–4.0 mEq/L	20 mEq/h IV[a]	
4.0–5.5 mEq/L	10 mEq/h	
>5.5 mEq/L	No potassium needed	

Abbreviation: IV, intravenous.

[a] Can use two peripheral lines or a central line if there is a need for more than 10 mEq/h. All of these patients must be on a cardiac monitor.

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Table 2
Insulin drip dosing for treatment of DKA

Regular insulin	Initial dose is 0.1 U/kg/h IV. Some institutions recommend a bolus of 0.1 U/kg and then the start of the infusion. Increase the insulin infusion by 0.05–0.1 U/h if the glucose is not decreasing or the anion gap is not closing (may require glucose infusion to maintain glucose >250 mg/dL). Decrease the insulin infusion if the glucose level is falling too quickly. The insulin infusion should remain at least 0.05 U/h to ensure that ketosis does not restart.
SQ fast-acting insulin (lispro)	Initial dose is 0.15 U/kg. Repeat every 2 h if glucose >250 mg/dL. Repeat every 4 h if glucose <250 mg/dL.

because this achieves a therapeutic level quicker. **Table 2** provides insulin dosing recommendations.

It is extremely important that the insulin infusion or SQ dosing not be stopped until the following criteria are met:

- Anion gap has closed: This marks the resolution of ketoacidosis, when SQ or IV insulin is required.
- Acidosis resolved: Bicarbonate level greater than 18 mEq/dL. This ensures the patient was not just converted to NAGMA and that the acidosis is truly resolved. Patients with a closed gap and bicarbonate less than 18 mEq/dL may need bicarbonate supplementation.

Once these criteria are met, insulin is transitioned from IV to long-acting SQ insulin. Patients with known diabetes who are already on long-acting insulin can receive their normal home dose. For new patients with diabetes, one calculates the total daily dose of insulin. **Box 1** provides dosing recommendations.[33] The patient receives half of their total daily dose as long-acting insulin and the rest as a sliding scale of insulin to accommodate meals. Continue the insulin infusion for 2 hours after the long-acting insulin is given to ensure it is metabolically active. After 2 hours, the insulin infusion is shut off. Glucometer checks are spaced to every 4 hours, and the frequent laboratory checks are stopped. The patient should be placed on a consistent carbohydrate diet. If a new patient with diabetes, the patient should have arrangements made to be seen by endocrinology and a dietician so that they can receive proper diabetic teaching.

Evaluation for Triggers

As part of the treatment, it is imperative to remember to treat any potential triggers that caused the patient to go into DKA, such as an infection.

Box 1
Total daily dose of insulin and long-acting insulin dosing[33]

Glargine

Total daily dose = 0.5–0.8 U/kg/d

Administer 50% of total daily dose as glargine

Remaining insulin can be divided as aspart for meal coverage

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PEARLS

- Patients presenting with a WBC greater than 25,000 should be evaluated for an underlying active infectious process.
- An increased or normal sodium concentration is indicative of profound free water loss.
- Hypokalemia on initial laboratory studies represents a severe total body depletion of potassium and requires careful and emergent replacement.
- Consider EDKA in patients who have metabolic acidosis and concurrent risk factors.

PITFALLS

- Failing to recognize an inciting or precipitating event in the development of DKA.
- Depending on hyperglycemia, AGMA, and ketonemia to all be present when diagnosing DKA.
- Not having a high level of suspicion for EDKA in patients on SGLT-2 or those with a history of diabetes, starvation, or perioperatively.

DISPOSITION

Most patients who present to the ED in DKA require admission to the hospital, with frequent and close monitoring of laboratory abnormalities and mental status.[34] The ultimate destination depends on the intensive care unit or intermediate care unit capability of the hospital. Patients in DKA should be admitted to an intensive care unit/intermediate care unit that can handle frequent laboratory studies, hourly glucometer checks, and the administration of electrolyte supplements and insulin. With national boarding issues, it is not unusual that these patients are managed in the ED until the anion gap is closed and they are transitioned to SQ insulin. These patients are admitted to a medical/surgical ward with every 4-hour glucometer checks. Most, if not all, are admitted. Some patients without infectious or other medical triggers are discharged home if they are considered highly reliable and have a plan that promotes the best possible chance of adherence so they do not have a recurrence. Insulin-dependent patients who do not or cannot adhere to insulin are at higher risk of recurrent episodes of DKA.[35] Patients on SGLT-2 should have close follow-up and monitoring by their primary care providers with strict information on the signs, symptoms, and triggers of DKA.[27,36]

SUMMARY

DKA continues to be a prevalent complication of diabetes that carries high morbidity and mortality. The diagnosis is made through careful history, physical examination, and recognizing inciting events that precipitate DKA in patients with diabetes. Appropriate laboratory and imaging work-up should be initiated promptly in addition to treatment with fluid hydration, electrolyte replacement, and insulin. Some patients may be suitable for discharge home from the ED, but many, if not most, require further resuscitative efforts and medical management in the hospital.

DISCLOSURE

The authors have no financial disclosures or commercial or financial conflicts of interest.

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